

Aptamers recognize glycosylated hemagglutinin expressed on the surface of vaccinia virus-infected cells

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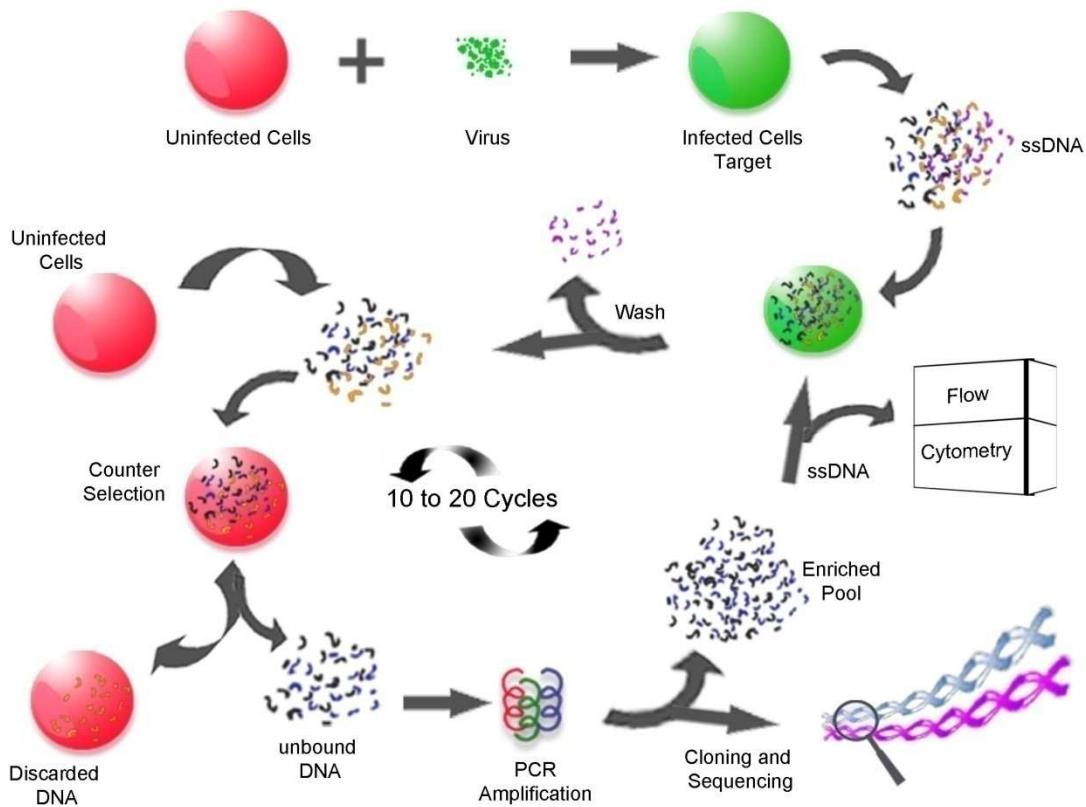


Figure S1: Cell-SELEX schematic using HeLa cells infected by VV WR as the target and uninfected HeLa cells for counter selection. After 20 rounds of selection, the DNA pool was enriched for the target cells, cloned, and sequenced to reveal the identity of the aptamers.

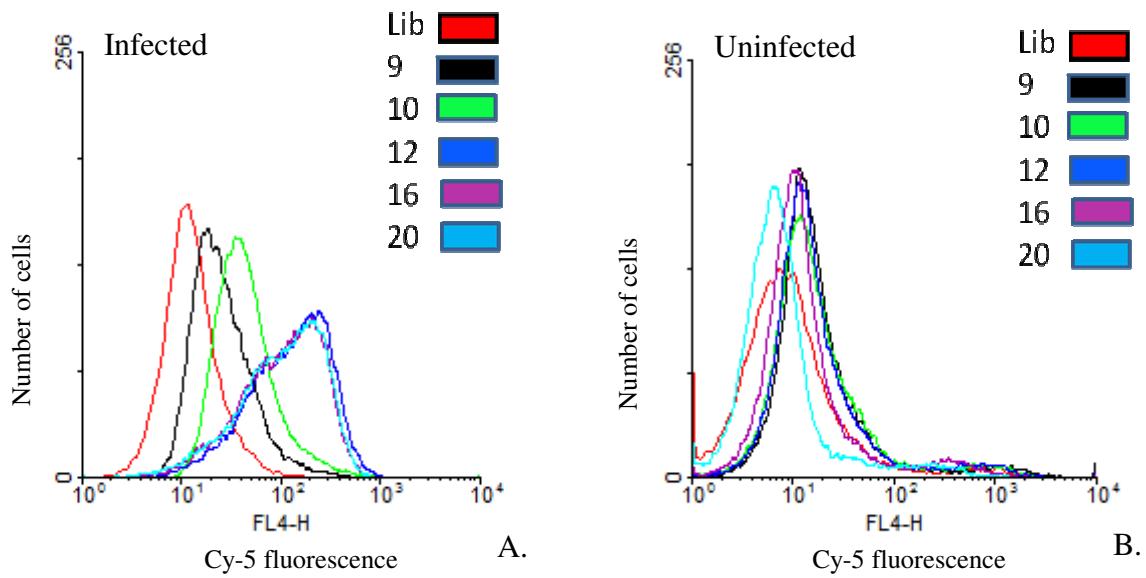


Figure S2. Enrichment of DNA pools observed using flow cytometer to monitor the progress of SELEX.

(A) A slow enrichment of the aptamer pools is observed as a shift on the x-axis caused by the Cy-5 fluorescence signal from pool 9 to 20 with VV WR-infected cells; (B) The enriched aptamer pools do not bind uninfected HeLa cells.

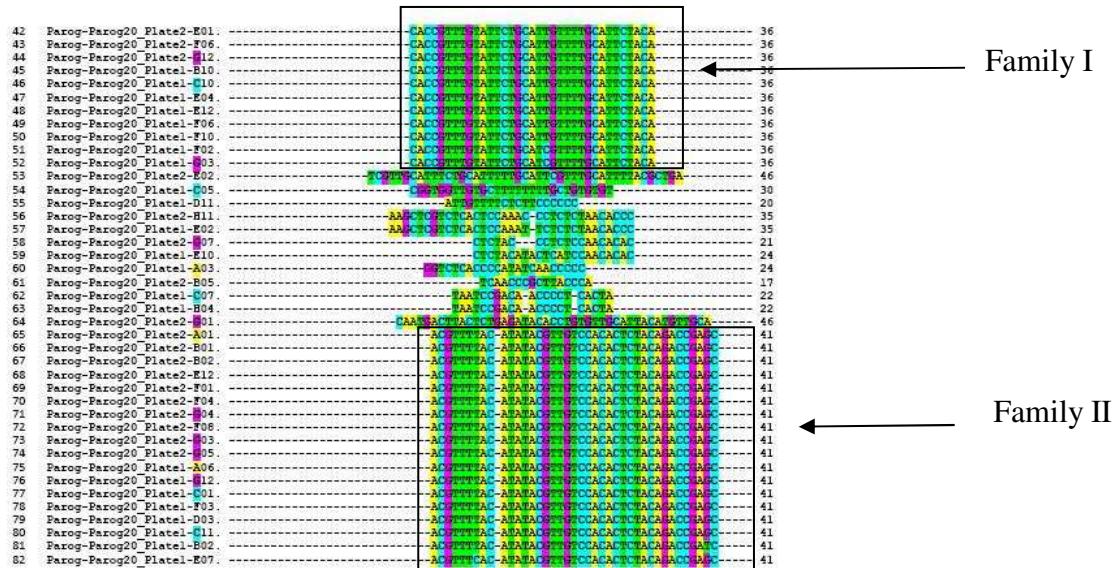
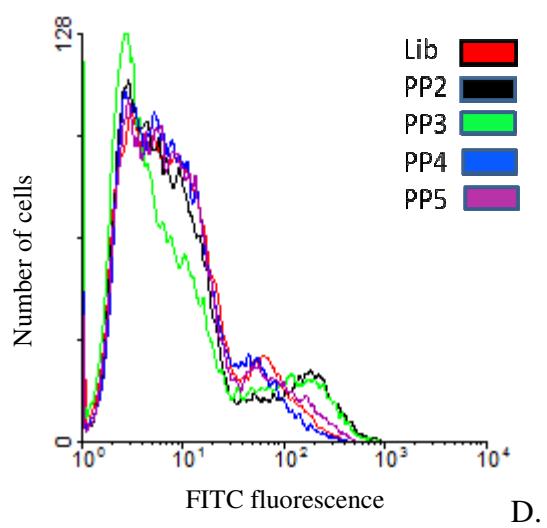
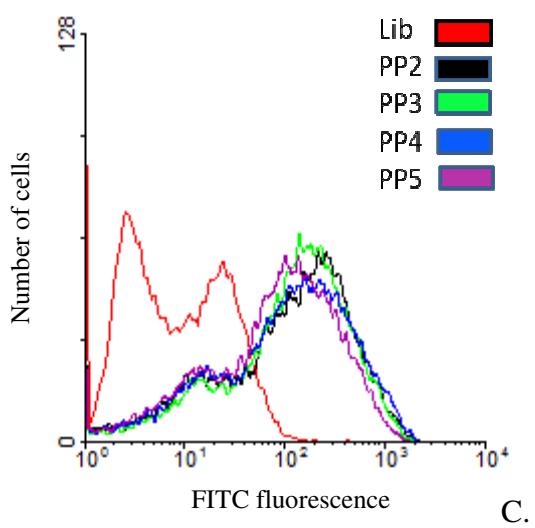
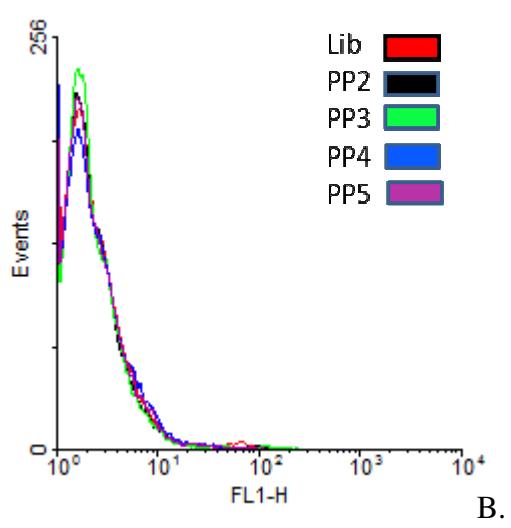
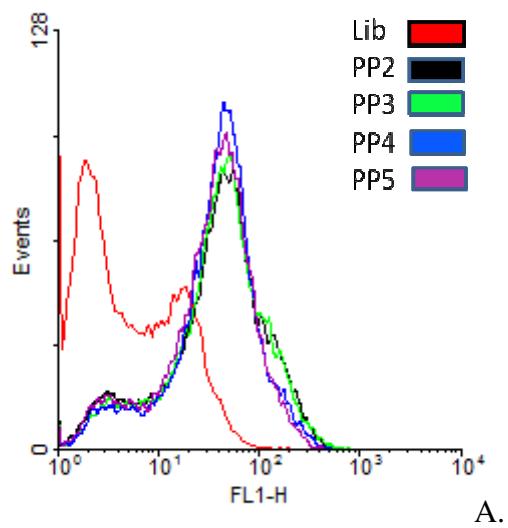


Figure S3: Alignment of aptamer sequences using Clustal X alignment software to classify the sequences obtained into families based on the number of repeats in each family. A few of the sequences were selected for further characterization.



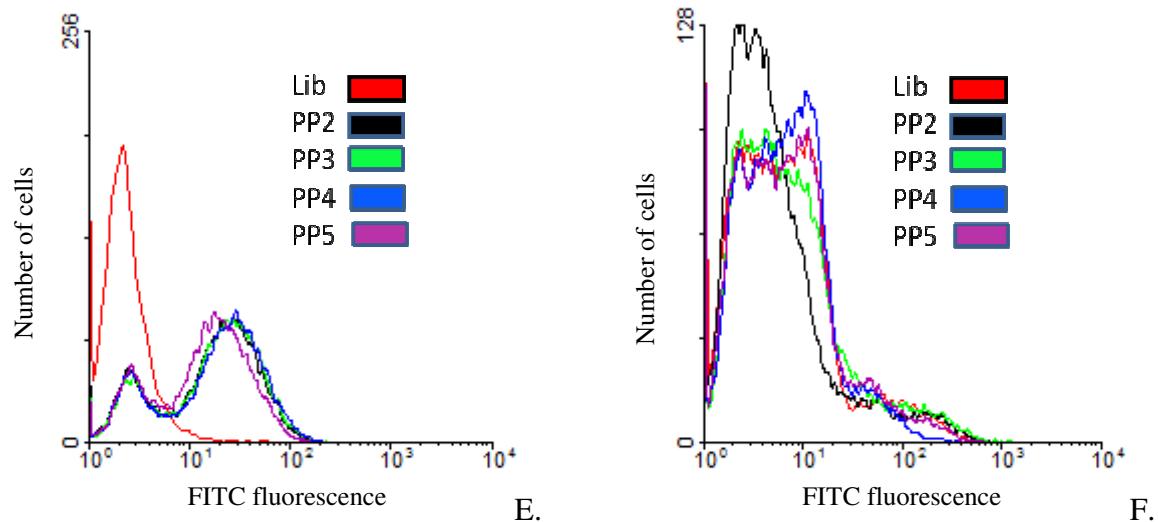


Figure S4: Aptamers can recognize different cell lines, including CV-1, RK13 and PK15 cells, infected with VV WR. (a) Aptamers bind CV-1 cells infected with VV WR, but (b) do not bind uninfected CV-1 cells. (c) VV WR-infected RK13 cells are recognized by aptamers, but (d) do not bind uninfected RK13 cells. (e) Aptamers bind PK-15 cells infected with VV WR, but (f) do not recognize uninfected PK-15 cells. Thus the selected aptamers recognize a virally encoded target.

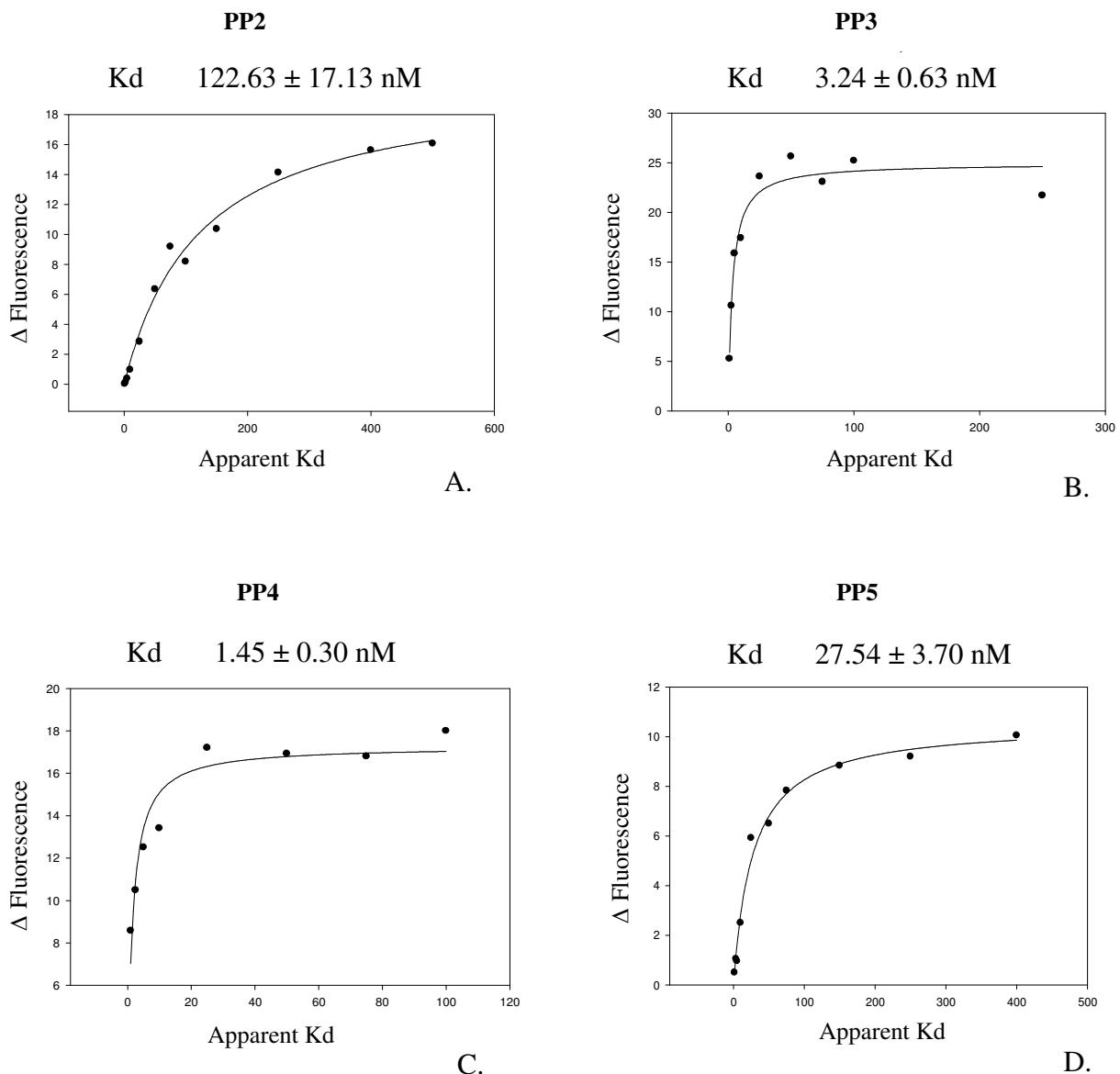


Figure S5. Apparent dissociation constants of the aptamer - target interaction were calculated by measuring the difference in mean fluorescence intensity of the aptamer and random DNA library bound on the surface of VV WR-infected HeLa cells with a flow cytometer and SigmaPlot 11.0 statistical software (A) Aptamer PP2; (B) Aptamer PP3; (C) Aptamer PP4; and (D) Aptamer PP5